Clinicopathological Study of Dysfunctional Uterine Bleeding in Postmenopausal Women

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Summary

There were 28 cases of postmenopausal dysfunctional uterine bleeding giving the incidence to be 5.38%. the duration of menopause and endometrial bleeding was statistically not significant. The hyperplastic endometrium was the commonest presentation in 39.29% of cases.

Introduction

Bleeding from the genital tract occurring after the menopause is much more sinister than premenopausal bleeding (Jeffocoate, 1981). Dysfunctional uterine bleeding (DUB) can develop in about 10% of cases of postmenopausal bleeding (PMB) (Dawn, 1997). PMB is the only common clinical indication of presence of endometrial cancer. Understanding the postmenopausal endometrium and its risk for developing carcinoma is important (Fortier, 1986). A clinicopathological study was done between various patterns of uterine bleeding with duration of menopause and endometrial histology.

Material And Methods

The study was carried out in the Department of Obstetrics and Gynaecology at MGIMS, Sevagram in cases of DUB.

The cases who were admitted as DUB were analysed. Amongst 520 cases of DUB there were 28 cases of PMB, all of whom had undergone dilatation and curettage and hysterectomy when needed. An attempt was made to correlate menstrual abnormalities with ovarian and endometrial histopathology in these cases.

Observations and Discussion

The incidence of PMB was 5.38% in the present study. Joshi and Deshpande (1964), Nayak et al (1976), Sagar (1980) and Dawn (1997) had reported postmenopausal bleeding in 0.54, 12.9, 9.96 and 10% cases respectively in dysfunctional uterine bleeding group.

Hyperplastic endometrium was commonest in 11 (39.29%) followed by proliferative endometrium in 5 (17.86%) as shown in Table I. A very high incidence of proliferative endometrium in 53.06% was reported by Sagar (1980). Previously it was believed that hyperplasia could rarely persist for more than a year or two after menopause but the concept of postmenopausal steroid function by adrenal, ovarian and other sites could lead to hyperplasia.

Table I
Postmenopausal Bleeding and Endometrial Histopathology

Endometrial patterns	No. of cases	Percentage
Proliferative	5	17.86
Secretory	2	07.14
Hormonol imbalance	3	10.71
Hyperplasia	11	39.29
Endometritis	1	03.57
No endometrium	6	21.42
Total	28	100

The proliferative and also hyperplastic endometrium indicate effects of oestrogen. As ovarian histopathology did not suggest stromal cell or granulos cell hyperplasia in all 28 cases , it suggests that extrauterine source of oestrogen such as adrenal or other site could be present. No endometrium was obtained in 21.42% of cases of PMB. Sagar (1980) had reported atrophic endometrium in 6.12% cases. This could be due to complete atrophy of the endometrium. The incidence of atrophic endometrium in association with DUB varies from 1.9 to 21.9%. Atrophy of endometrium may be associated with large dilated venules situated superficially under a thin endometrium. These venules may rupture and are probably the commonest cause of postmenopausal bleeding (Davey, 1995). Selective atrophy of endometrium could be explained by the failure of uterine receptors to respond to the oestrogenic

stimulus (Bourne and Williams, 1962). Secretory endometrium found in 7.14% cases of PMB could be due to occasional ovulation in such cases (Jeffcoate, 1981).

Of 28 cases of PMB a maximum of 9 (32.14%) had sought medical help between 1-2 and after 5 years of attaining menopause. Of all cases of PMB 16 (57.14%) cases had presented with continuous bleeding and 7(25%) cases with menorrhagia. Table II shows that, statistically the bleeding pattern in postmenopausal bleeding in relation to duration of menopause was found to be insignificant in present series ($p>0.05$). Available literature did not show such type of study.

Table — II
Different Patterns of Bleeding with duration of Menopause

Duration of Menopause	Meno-rrhagia	Polymeno-rrhagia	Polymeno-rrhagia	Metro-	Cont. Bl orrhoea	Amen	Total
1	-	-	-	-	3 (10.71)	-	3 (10.71)
1-2	1 (3.57)	-	-	1 (3.57)	7 (25)	-	9 (32.14)
2-5	2 (7.14)	-	-	1 (3.57)	2 (7.14)	2 (7.14)	7 (25)
> 5	4 (14.28)	1 (3.57)	-	-	4 (14.28)	-	9 (32.14)
Total	7 (25)	1 (3.57)	-	2 (7.14)	16 (57.14)	2 (7.14)	28 (100)

$p>0.05$

Figures in parenthesis denotes percentage.

Correlation of ovarian histopathology with PMB (Table III) could be done in 16 cases, which showed that 57 % of DUB had positive ovarian pathology. Stromal cell hyperplasia was present in 32.25%, serous cyst in 18.75% and granulosia cell hyperplasia in 6.25%. Cortical stromal hyperplasia of ovaries at menopause is due to stimulation by pituitary gonadotrophins even after menopause(Mitra, 1964). The study of Boss et al (1965) had reported that 28% of ovaries obtained at autopsy of postmenopausal women showed stromal cell hyperplasia which was unrelated to hormone production. In stromal cell hyperplasia the transitional cells of ovarian cortex produce steroid hormone.

Table III
Postmenopausal Bleeding and Ovarian Histopathology

Ovarian histopathology	No.	%
Granulosa cell hyperplasia	1	06.25
Stromal cell hyperplasia	5	31.25
Serous cyst	3	18.75
Unremarkable	7	43.75
Total	16	100

The stroma is an androgenic compartment of ovary. Oestrogen changes when present may result from peripheral conversion of excessive androstenendione to oestrogen (Young and Scully, 1995). We believe this fact. We had associated serous cyst in 31.25% cases in PMB. Mitra (1964) had reported incidence of 25% of serous cyst in postmenopausal group.

Association of hyperplastic endometrium and endometrial carcinoma is well known. Our finding of higher percentage of hyperplastic endometrium suggests that all cases of PMB must undergo traditional methods of dilatation and curettage to rule out malignancy and establish plan of management.

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